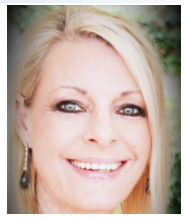
15 LEADING MISPERCEPTIONS ABOUT BARIATRIC SURGERY AND OBESITY

There are some mistaken beliefs that exist about bariatric surgery and obesity. Here are 15 common ones.



Prof Tess van der Merwe
CEO CEMMS (SA), Clinical Researcher Waterfall City Hospital and UP, Chair SASSO, Consultant World Obesity

1 The procedures are dangerous.

Bariatric surgery is performed as a laparoscopic procedure. In the CEMMS (SA) centres professionals are experienced with very advanced skills, at many levels. The national data base that is kept at the head office and training facility at Waterfall City Hospital, indicate that the mortalities for the country (nine accredited centres) are 0.4 %. Major surgical complications at 30 days are 3% (including difficult redo surgery), over 30 days, at as long as a 10 year follow up, it will be up to 8% over time. Medical complications post operatively is 4% and mostly from hypostatic pneumonias in the very obese patients.

These figures should be compared to outcomes of other surgery types with high technical difficulty grading eg colorectal surgery. Very few specialities will adhere to peer review scrutiny and the keeping of a data base, so stats on outcome is mostly unavailable to the South African market. Professionals performing bariatric surgery in unaccredited centres do not adhere to a national database.

2 The procedures are very expensive and will require a long recovery time.

Our database has shown that the patients electing bariatric surgery as a treatment modality would have been through a mean of four serious weight loss and weight regain cycles. This could have cost anything from R7 500 – R10 000 per cycle and is paid towards off label treatment strategies with no proven track record or scientific data on outcome. Over time, co-morbidities will increase. The mean amount of co-morbidities documented at baseline is six. The scheduled pharmaceuticals required to treat all these co-morbidities would include medication from a mean of eighjt drug classes. Since bariatric surgery is extremely effective at placing most co-morbidities in resolution, or complete/partial remission, the average amount of drug classes will decrease to 1.2 at seven years. Health economic return on capital investment is already apparent at 18 months[1]). Approximately 70% of the patients at Waterfall City hospital obtain medical aid funding from medical aids that support bariatric surgery. A 20% co-payment is levied and a certain plan type is a requirement.

The average surgical time for the most commonly performed procedure (LGBP) is approximately 45-60 minutes. Hospital stay could be one to three nights depending on the procedure selected. Work related off time is 10-14 days from the time of surgery in most cases. Most patients will be able to comfortably perform admin work from home, from the day they leave hospital. The liquid phase requires a somewhat more tranquil environment than the office.

3 The patients most suitable are morbidly obese.

Whilst the super morbidly obese patients often attract the most media attention and plight for compassion, it is the BMI group 30-48 that has the greatest representation for cases done in the national and international arena. This BMI group is also the group that is most frequently included in randomised controlled trials[2]. Outcome data for the lowest BMI category of 30-35 is well represented[3]. At Waterfall City Hospital the mean BMI of cases operated on is 44 +/-6. Around 30% of cases presenting for surgery will be diabetics at a BMI 30-40. Patients at a BMI 55 and above should be taken care of in the most experienced teams, as they often pose clinical and technical challenges that are unique and require the most expert of management. In addition, savings on health economic expenditure with weight loss will be the highest in patients at a BMI above 45 and greater in all diabetic vs non diabetic patients for every category of BMI.

4 The procedure is cosmetic.

It is very disturbing that this narrative and notion still exists within the realms of insurers, policy groups and certain medical aids. Rather there should be an understanding of this chronic disease, and policies need to be adjusted to incorporate the myriad of literature that supports the evidence of obesity as a chronic disorder of energy homeostasis[4]. Obesity is not a disease of passive accumulation of excess weight, but the strong biological defence of excess fat mass at the level of the ventromedial nucleus, arcuate nucleus, paraventricular nucleus and lateral hypothalamic area[5]. The mechanisms underlying this upward setting of the body weight set point are complex and multifactorial.

5 Obesity can be 'fixed' with enough will power or the right diet.

A recent scientific statement by the International Endocrine Society has clearly highlighted the pathogenesis of obesity as a brain-centric model in which the brain, by virtue of its strong hypothalamus and hind brain operational control of food intake and energy expenditure, imposes excess calories on the adipocyte. A very modest but persistent energy balance mismatch (1-3% more calories consumed than expended per year) can explain the slow but continuous accumulation of body fat over many years that are characteristic of most humans that are obese[6]. Although the acute increase of body fat is often reversible, incremental, sustained increase of body-fat mass typically ends up becoming part of the total body fat mass that is biologically defended. It is for this reason that the weight loss induced by diet or life style changes is often ultimately regained, even in the face of adherence to a more healthy diet or lifestyle. These observations imply that although the mechanism underlying the gradual increase in the defended level of body fat may have been triggered by one or more of the many environmental exposures, simple withdrawal of the offending exposure is unlikely to reverse the increased body fat once it becomes established. Instead the energy homeostasis system has been upwardly reset, so that the higher level of body fat is relatively resistant to lifestyle intervention.

6 Exercise will solve the problem.

Variations in energy intake typically have a much larger effect than variations in energy expenditure on overall energy balance. Therapeutic interventions that raise energy expenditure sufficiently to cause weight loss eventually trigger increased food intake as a compensatory response[7]. For this reason, the ability to manipulate energy intake is the more pressing goal in obesity treatment. In addition, although energy intake generally adjusts well to energy expenditure, with a tight coupling found in free living adults, this compensation at low levels of energy expenditure appears far less accurate and will be present in obese patients with a very sedentary life style[8]. Not with standing, the myriad additional and other beneficial effects of even modest training cannot be overstated.

7 Changing the macronutrients of your diet will solve the problem.

When calorie intake is held constant, body fat accumulation is not affected by even pronounced changes in the amount of fat vs carbohydrate in the diet. This raises the question: "Is a calorie a calorie?" Mechanism related to increased insulin secretion, nutrient partitioning, cellular starvation or other metabolic pathways have never been proven to be present when trials have been kept isocaloric. Furthermore, the hyperinsulinemia of obesity is typically associated with normal or elevated circulating glucose or fatty acid levels, a combination inconsistent with a state of insulin mediated cellular starvation, the latter being the main proposed theory of a higher weight loss on the high fat/high protein diet[9]. There is also little or no direct evidence that insulin's antilipolytic action is an independent determinant of fat mass[10]. From this there is inference that the effect of diet composition perse on metabolic variables suggested to contribute to obesity pathogenesis, do not play any clinical role unless they promote a direct increase in calorie intake eg sugary drinks.

8 The patients will be replacing one addiction with another one.

The concept of food and sugar addiction needs to be laid to rest. It is an outdated notion that has the simple goal of oversimplification of the complexity of dopamine and the role it plays in entrenching habit forming pathways[11]. It also does not consider the myriad of physiological and neuro-molecular mechanisms that have been identified in the integrative short and long term control of feeding behaviour. Available evidence points to a host of afferent humoral and neural signals arising from the interaction of food with the GI tract as primary determinants of the size of an individual meal (Hind brain circuits and the parabrachial nucleus).[12] In 1922, Sir Harvey Cushing as president of the International Endocrine society, advocated strongly in favour of adopting scientific method and abandoning empiricism to better diagnoses and treat endocrine diseases.

9 The patients will be better off doing it the 'natural way'.

Rather than viewing recovery of lost weight as evidence of non-compliance, patients and practitioners should view this phenomenon as an expected physiological response to weight loss. This immediately beckons the question of what is regarded as the 'natural way' of weight loss. Accepting that less than 5% of patients will sustain a 5kg weight loss over five years with lifestyle and

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<< dietary modifications, this can hardly be branded as a successful form of intervention of a chronic disease. In the same way as treating diabetes, hypertension and hypercholesterolemia would be unsuccessfully treated in most cases without medical intervention. The incorrect underlying narrative is that the disease of obesity is still being forced into the narrow minded box of a lifestyle disease, when it has for decades already been proven to be a chronic disease with a complex aetiology. The specialist medical fraternity needs to take ownership of the disease in a far more forceful manner and use scientifically successful strategies of treatment, with documented outcome data, to lead. Relying on exploitative, market driven, populist and pseudo-scientific forces (even amongst the medical profession) to lead this field of treatment, has paid no dividends in curbing the epidemic.

10 Diabetes will not be cured post-surgery.

No chronic disease can be cured. But it can be placed in remission, and this bariatric surgery can do to a much larger and more successful degree than medical treatment alone. The argument should not be for or against either form of treatment, but whether a combination of both surgical and medical treatment can be more effective in preventing long term complications and organ damage. It stands to reason that any length or degree of disease remission, achieved with bariatric surgery as a treatment strategy, can only be viewed as favourable and as part of improving quality of life. Fewer than 50% of the approximately 450 million type 2 diabetics can control their blood sugar levels adequately by diet, exercise and drug treatment. A randomised controlled trial by Mingrone *et al* showed that 0% of patients on medical treatment achieved remission at five years, compared to the 37% of patients in the laparoscopic gastric bypass group (LGBP), and 63% of the patients undergoing a biliopancreatic diversion and duodenal switch procedure (BPD-DS).[13] However, patients who relapsed after two years only required a single oral drug for control of their DM (ADA criteria), 47% of who required insulin prior to their bariatric surgery. Overall, 87% of surgery patients required no medication for hyperglycaemia by the end of five years. None required insulin. Degree of weight loss was not associated with diabetes remission or relapse, supporting the notion that bariatric surgery activates weight independent mechanisms of diabetes control. It also suggests that differences in surgical anatomy explain variances in clinical effectiveness of surgical procedures. The BPD-DS patients have a larger gastric reservoir than those who undergo LGBP. This finding is suggestive that intestinal mechanisms exert a more important anti-diabetic effect than would changes in gastric volume, and would also explain why the lone sleeve gastrectomy is a poor choice for diabetes remission[14]. Various secondary outcome measures to assess cardio metabolic risk also showed a far superior outcome in the surgically treated group of patients[15]. Overall drug use to control co-morbidities in the surgical patients decreased, compared to an increase in the medically managed group, over time[16]. Notably, LGBP patients tend towards a very high HDL-Cholesterol, the mechanism of which is still unclear.

11 The patients will lead an abnormal life.

It would be far more accurate to argue that patients would go from leading a life with abnormal physiology, to progressing to a normal physiology. In addition, quality of life markers in patients undergoing bariatric surgery are far better than compared to medically managed patients. Repeated implementation of the RAND36-Item Health survey that includes quality-of-life measures, indicated that bariatric surgery patients score significantly better than medically treated patients or all the subdomains (physical functioning, bodily pain, physical activity, vitality, emotional stability, general health, social functioning and mental health), as well as on the overall total score.[17] The narrative of a severely restricted, abnormal lifestyle is a misnomer to the extreme.

12 The sleeve gastrectomy will not require vitamin replacement.

The European Association for the study of Obesity published the 2017 recommendations for post-bariatric surgery medical management[18]. It is clear that patients with a Sleeve Gastrectomy will require iron ferrumate and vitamin B12 replacement to the same degree as what is required for LGBP. In addition, it is recommended

that folic acid be taken a part of a MVT. It is also prudent to replace Vitamin D and calcium as calcium-citrate, as intake will be restricted. These nutrients will often be deficient at onset. Regular PTH measurements should be used as an indicator of adequate replacement. The misperception amongst laparoscopic surgeons that the follow up treatment of the patients undergoing sleeve gastrectomy, will be less labour intensive and need to be addressed with vigour. The notion of selling it as a safer procedure is also unfounded, as complications may be more sinister and difficult to handle. Long term outcome data is not as good or even comparable as for LGBP or BPD-DS.

13 Patients with depression or eating disorders do not qualify.

Patients with reactive, endogenous and bipolar depression do well with LGBP and BPD-DS procedures. As a general rule lithium needs to be replaced pre-operatively with another drug regimen, as a more drastic blood level variation and toxicity can be expected. In addition, the absorption of XL formulations does not fare well and should be curtailed.

Wellbutrin has been noted to induce more anxiety post operatively. Binge eating disorder frequently improves dramatically after LGBP. Night eating disorder must receive optimal care prior to surgery, particularly when it forms a part of uncontrolled bipolar disease. Patients with excessive and severe sweet eating disorders have better success rates with the BPD-DS, with very good outcomes. It is strongly recommended that the patients with mental and eating disorders be taken care of peri-operatively by a psychiatrist that is familiar with the different procedures and functions as a part of the CEMMS team.

14 The procedure should only be performed in adults.

A prospective trial of 242 obese adolescents undergoing bariatric surgery indicated significant improvements in weight, cardio-metabolic health and weight related quality of life three years after surgery.[19] This publication in the *New England Journal of Medicine* in 2017, was part of an era whereby several other investigators produced similar results. At three years, remission of type 2 diabetes was 95%, remission of abnormal kidney function occurred in 86%, remission of pre diabetes occurred in 76%, remission of hypertension in 74% and remission of dyslipidaemia in 66%. Adolescents of 13-19 were included and had a BMI 40 or above. The risks of major complications at 30 days were equal to that observed in adults. At Waterfall City Centre the major complication rate for the adolescent cohort is less than 1%. In view of a relatively higher risk of iron and B12 deficiency, it is recommended that these adolescents only be operated on at the most established of centres, where an endocrinologist will form a part of the care. That being said, they have been found to be a particularly successful and gratifying group of patients with an unusually unburdened tolerance of the procedure. Both physically and mentally.

15 Surgery is a quick fix for patients who do not want to do it the 'hard way'.

In my 30 years of dealing with obesity and its management, this is probably the statement that has been representative of the greatest degree of total lack of insight. Lack of insight into the disease of obesity, its pathogenesis, its evolution, its research and its complexity. Proponents of the 'natural and hard treatment route' (whatever that may mean), will likewise be uttering sentences that resemble the following: 'bariatric surgery is expensive' and 'dangerous' and 'will never curb the epidemic'. The reality is that many drugs are dangerous but advocated; many expert treatment options for other chronic diseases are expensive but have a modest if not minimal outcome effect. Bariatric surgery was never designed to curb an epidemic, in the same manner as insulin was never designed to curb the flood of diabetes. Unless we can, with a sense of urgency, rephrase the narrative that is used when addressing obesity, whether it is by the lay public, the media or within the medical fraternity itself, the hope that we will be treating these patients with the dignity and scientific correctness they deserve will remain elusive. MC

References available on request